

**RANDOMIZED CONTROLLED TRIAL ON EFFECT  
OF INTRAPERITONEAL BUPIVACAINE  
INSTILLATION ON POST OPERATIVE PAIN  
RELIEF IN LAPAROSCOPIC SURGERY**



**Dissertation submitted in partial fulfillment of regulation for the  
award of M.S. Degree in General Surgery  
(Branch I)**



**THE TAMILNADU  
DR. M.G.R. MEDICAL UNIVERSITY  
Chennai  
March - 2010**

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Coimbatore - 641 014  
March - 2010**

## **CERTIFICATE**

**Certified that this is the bonafide dissertation done by  
Dr.C.RAMAMURTHY and submitted in partial fulfillment of the  
requirements for the Degree of M.S., General Surgery, Branch I  
of The Tamilnadu Dr. M.G.R. Medical University, Chennai.**

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## **DECLARATION**

I solemnly declare that the dissertation titled **“RANDOMISED CONTROLLED TRIAL ON EFFECT OF INTRAPERITONEAL BUPIVACAINE INSTILLATION ON POST OPERATIVE PAIN RELIEF IN LAPAROSCOPIC SURGERY”** was done by me from 2007 onwards under the guidance and supervision of **Prof. Dr. A. RAMAMOORTHY M.S and Prof. Dr.G.Mohan M.S**

This dissertation is submitted to the **Tamilnadu Dr. MGR Medical University** towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

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## ETHICS COMMITTEE

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Period of Study : 2007 - 2010  
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Dissertation Topic : Effect of intraperitoneal  
bupivacaine on post-operative pain relief in  
laparoscopic procedures.  
The Ethics Committee, Coimbatore Medical College has  
decided to inform that your Dissertation is accepted /  
~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to  
proceed with the above Study.

Coimbatore - 14.

Date : 13.02.08

*K. N. N. N. N.*  
Secretary  
Ethics Committee

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I would like to thank my **Asst Prof. Dr.G.Ravindran,.** for his consistent encouragement and support.

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I extend my heartfelt thanks to all the **Patients** who co-operated for this study.

## **LIST OF ABBREVIATIONS**

<b>VAS</b>	<b>:</b>	<b>Visual Analog Scale</b>
<b>NSAID</b>	<b>:</b>	<b>Non Steroidal Anti Inflammatory Drugs</b>
<b>POST.OP</b>	<b>:</b>	<b>Post Operative Period</b>
<b>BMI</b>	<b>:</b>	<b>Body Mass Index</b>
<b>ASA</b>	<b>:</b>	<b>American Society of Anesthesiologists</b>
<b>CO2</b>	<b>:</b>	<b>Carbon – di oxide</b>
<b>BUPIVACAINE</b>	<b>:</b>	<b>Bupivacaine hydrochloride</b>
<b>AR</b>	<b>:</b>	<b>Analgesic request rate</b>
<b>AC</b>	<b>:</b>	<b>Analgesic Consumption</b>
<b>NCA</b>	<b>:</b>	<b>Nurse controlled Analgesia</b>
<b>LNCAR</b>	<b>:</b>	<b>Latency of the First Nurse Controlled Analgesia Request</b>

## ABSTRACT

**Background:** Although pain after laparoscopic surgery is less intense than open surgery, some patients still experience considerable discomfort. The aim of this project is to study the influence of intraperitoneal instillation of bupivacaine on pain after laparoscopic surgery, since there is some controversy in recent trials.

### Methods

In this study 120 patients qualified for laparoscopic abdominal surgery were randomized to four groups. Group A received 2mg/ kg of bupivacaine in 60ml of normal saline immediately after creation of pneumoperitoneum. Group B received 2mg / kg of bupivacaine in 60ml of normal saline at the end of planned surgical procedure. Group C received 60 ml of normal saline immediately after creation of pneumoperitoneum. Group D received 60ml of normal saline immediately after creation of pneumoperitoneum. Group D received 60ml of normal saline at the end of planned surgical procedure. The patients with ASA grade III or more and patient who had hypersensitivity to bupivacaine were excluded from the study.



The primary end points of the study were post operative pain intensity on a visual analog scale and incidence of shoulder tip pain. The secondary end points includes the latency of nurse – controlled analgesia activation, the analgesia request rate and analgesic consumption.

## Results :

Significantly lower visual analog scores were observed in group A versus group C and B versus D during the initial 24 and 48 hr. respectively. The patients in group A versus group B reported significantly lower pain at 4 hour ( $p < 0.001$ ) and 8h ( $p = 0.003$ ) post – operatively, but the difference was not significant after 12,24 and 48 hour. None of the group A patients reported shoulder tip pain, whereas it was reported by 3 patients in group B, 6 patients in group C, 7 Patients in group D ( $P < 0.01$ ). The latency of nurse controlled analgesia activation was  $426.8 \pm 57.2$  min. Group A as compared with  $307 \pm 39.8$  min in group B,  $109.3 \pm 51$  in group C and  $109 \pm 46.5$  min in group D ( $p < 0.001$ ). A significantly lower analgesia request rate was observed in group A versus C, as compared with group B versus D, throughout the entire study period ( $p < 0.05$ ).

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# INTRODUCTION

Although laparoscopic surgery is associated with less pain than contemporary open procedures, it is definitely not pain free and the magnitude of postoperative shoulder and abdominal pain in the early postoperative period is still quite significant. This postoperative pain is a major concern not only for the patients, but also healthcare workers; and it often contributes to overnight hospital stay after this minimally invasive surgical procedure.

The severe pain we encountered in the laparoscopy patients during their post operative period may be secondary to tissue injury. One possibility is peritoneal irritation largely due to 1) CO<sub>2</sub> pressure in the abdomen, 2) blood left in the abdomen after surgery, and 3) diaphragmatic irritation. A higher intraperitoneal CO<sub>2</sub> pressure has been shown to generate more intense pain compared with a lower one, and laparoscopic procedures done without CO<sub>2</sub> insufflation at all are associated with less pain. In addition, surgery closer to the diaphragm has been shown to cause more pain than that following manoeuvres at lower sites in the abdomen. Another possible source of pain in the laparoscopy patients is pain from sustained intraoperative pressure on

capillary beds in the abdominal and possibly retroperitoneal viscera, causing nociception but resolves over the first few postoperative hours.

Intraperitoneal instillation of local anaesthetics at the time of surgery to control pain after laparoscopic surgery has been extensively studied in numerous randomized trials and found to have conflicting results. To answer this question, we designed a prospective randomized controlled trial with the specific aim to study the analgesic effect of intraperitoneal bupivacaine in the postoperative setting after laparoscopic surgery. If we can improve pain control after this minimally invasive procedure, it might result in decreased postoperative requirement of narcotic ,NSAIDS analgesia and its associated side-effects. It may also result in early recovery and the same day discharge of the patients with significant cost-containment for the patient and healthcare systems in future.

In the future remote handling technology will overcome the manipulative restriction in the current instruments thereby indirectly reduce the post operative pain.

## AIM

- To study the post operative analgesic effect of intraperitoneal bupivacaine instillation in laparoscopic surgery.
- To compare the analgesic effect of intraperitoneal bupivacaine instillation immediately after the creation of pneumoperitoneum and at the end of planned surgical procedure.

# REVIEW OF LITERATURE

## HISTORY OF LAPAROSCOPIC SURGERY

1901 :

The first experimental laparoscopy was performed in Berlin by German surgeon Georg Kelling,

1910

H.C. Jacobaeus of Stockholm published a discussion of the inspection of the peritoneal, pleural and pericardial cavity.

1911

Bertram M. Bernheim, from Johns Hopkins Hospital introduced first laparoscopic surgery to the United States. He named the procedure of minimal access surgery as "organoscopy". The instrument used was a proctoscope of a half inch diameter and ordinary light for illumination was used.

1911

H.C. Jacobaeus, again coined the term "laparothorakoskopie" after using this procedure on the thorax and abdomen.



1918

O. Goetze, developed an automatic pneumoperitoneum needle characterized for its safe introduction to the peritoneal cavity.

1920

Zollikofer of Switzerland discovered the benefit of CO<sub>2</sub> gas to use for insufflation, rather than filtered atmospheric air or nitrogen.

1929

Kalk, a German physician, introduced the forward oblique (135 degree) view lens systems. Goetze of Germany first developed a needle for insufflations.

1929 (2)

Heinz Kalk, a German gastroenterologist developed a 135 degree lens system and a dual trocar approach.

1934

John C. Ruddock, an American surgeon described laparoscopy as a good diagnostic method, many times, superior than laparotomy. John C. Ruddock used the instrument for diagnostic laparoscopy which consisted a built-in forceps with electro coagulation capacity.

1936

Boesch of Switzerland is credited for doing the first laparoscopic tubal sterilization

1938

Janos Veress of Hungary developed a specially designed spring-loaded needle, called veress needle.

1939

Richard W. Telinde, tried to perform an endoscopic procedure by a culdoscopic approach, in the lithotomy position.

1939

Heinz Kalk published his experience of 2000 liver biopsies performed using local anaesthesia without mortality.

1953

The rigid rod lens system was discovered by Professor Hopkins. The credit of videoscopic surgery goes to this surgeon who has revolutionized the concept by making this instrument.

1960

Kurt Semm, a German gynaecologist, who invented the automatic insufflator.

1960

British Gynaecologist Patrick Steptoe adapted the techniques of sterilization by two puncture technique.

1972

H.Coutnay Clarke first time showed laparoscopic suturing technique for hemostasis.

1973

Gaylord D. Alexander developed techniques of safe local and general anaesthesia suitable for laparoscopy.

1977

First Laparoscopic assisted appendicectomy was performed by Dekok. Appendix was exteriorized and ligated outside.

1977

Kurt Semm first time demonstrated endoloop suturing technique in laparoscopic Surgery.

1978

Hasson proposed a blunt mini-laparotomy which permits direct visualization of trocar entrance into the peritoneal cavity.

1980

In United Kingdom Patrick Steptoe, started to perform laparoscopic procedures.

1983

Semm, a German gynaecologist, performed the first laparoscopic appendicectomy.

1985

The first documented laparoscopic cholecystectomy was performed by Erich Mühe in Germany in 1985.

1987

Ger reported first laparoscopic repair of inguinal hernia using prototype stapeler.

1987

Phillipe Mouret, has got the credit to perform the first laparoscopic cholecystectomy in Lyons, France using video technique. Cholecystectomy is the laparoscopic procedure which revolutionized the general surgery.

1988

Harry Reich performed laparoscopic lymphadenectomy for treatment of ovarian cancer.

1989

Harry Reich described first laparoscopic hysterectomy using bipolar dessication; later he demonstrated staples and finally sutures for laparoscopic hysterectomy.

1989

Reddick and Olsen reported that CBD injury after laparoscopic cholecystectomy is 5 times that with conventional cholecystectomy.

1990

Bailey and Zucker in USA popularized laparoscopic anterior highly selective vagotomy com1994, A robotic arm was designed to hold the telescope with the goal of improving safety and reducing the need of skilled camera operator.

1996

First live telecast of laparoscopic surgery performed remotely via the Internet. (Robotic Telesurgery).

## NOCICEPTIVE PAIN

*Nociceptive pain is caused by an injury to body tissues.*

Nociceptive pain occurs as a result of the activation of the nociceptive system by noxious stimuli that can cause mechanically, chemically, or thermally induced damage to tissue integrity. The nociceptive system originates in peripheral tissues, spans the spinal cord, traverses the brain stem and thalamus, and terminates in the cerebral cortex, where the sensation of pain is perceived. Peripheral tissues are innervated by nociceptors, highly specialized primary sensory neurons, which contain specific receptors or ion channels at their peripheral terminals. Activation of these receptors or ion channels by noxious stimuli generates depolarizing current (or action potential or electrical impulse) which is then relayed to the brain for pain perception

## NOCICEPTORS AND AFFERENT NERVES

High threshold receptors, probably free nerve endings.

There are several distinct types of Nociceptors:

- Mechano-Nociceptors - generally stimulus specific (intense pressure, pinching); mediate fast/first pain, via AS (small diameter, myelinated) afferent fibres

- Polymodal Nociceptors - respond to many different forms of noxious stimuli: chemicals, intense heat, etc.; mediate slow/second pain, via C (small diameter, unmyelinated) afferent fibres
- '*Silent*' nociceptors - respond only after tissue damage (or sensitization)

Nociceptors also differ in their speed of conduction and in their capacity to be sensitized during inflammation, injury, and disease. The A-delta fiber and the C-fiber nociceptors are the 2 main classes of nociceptors. C-fibers are the most common; about 70% of all nociceptors are of the C-fiber type. Their axons are unmyelinated and their cell bodies are small. When activated, C-fibers conduct action potentials slowly, resulting in prolonged burning pain. A-delta fibers have thinly myelinated axons with medium- to large-diameter cell bodies. When activated, they conduct impulses at a fast rate and produce sharp, pricking pain.<sup>7</sup>

The nociceptive pain pathway consists of 4 processes: *transduction*, *conduction*, *transmission*, and *perception*.<sup>1</sup> Nociception begins with the activation of the specific receptors or ion channels in the peripheral

terminals of nociceptors by noxious stimuli. The activated receptors convert the noxious stimuli into electrical current, a process called *transduction*. The current generated at the peripheral terminals of sensory fibers depolarizes the nociceptor membranes generating action potentials. The latter are *conducted* along the nociceptor axons to their cell bodies, located in the dorsal root ganglion (DRG) in the spinal cord, and then to their central terminals located in the dorsal horn. Here, the action potentials initiate neurotransmitter release from nociceptor central terminals which relay the signal across synapses (*transmission*) to the dorsal horn neurons. The signal is then relayed via ascending nociceptive pathways to higher centers in the brain where it is perceived as pain (*perception*).<sup>5</sup>

## Gate Control Theory

This was proposed in an attempt to overcome the limitations of a specific and direct neural pathway serving pain and nociception.

The basic concept is that signals elicited in afferent neurons by noxious stimuli can be blocked or filtered by a synaptic 'gate' in the dorsal horn of the spinal cord. The story is complex and there are many controversial features. However, the principle does seem to work in practice, even if the precise mechanisms are unsettled.



The 'gate' is believed to be located in the substantia gelatinosa (SG) of the dorsal horn. Neurons of the SG make connections with the terminals of primary afferent fibres and also the dendrites of dorsal horn cells. Through either pre-synaptic or postsynaptic inhibition, the substantia gelatinosa neurons appear to be able to block (or reduce) activation of second order neurons by nociceptive inputs; these 'gating' effects of the SG neurons can be activated by:

(a) inputs in large diameter (A) afferents innervating the injured area.

(This provides the basis for pain relief by selective activation of large diameter afferents, as in transcutaneous electrical nerve stimulation - TENS).

(b) activation of neurons in certain brainstem regions, which send axons to the spinal cord.

## CNS Pathways

Nociceptive afferents synapse with two main groups of neurons in the dorsal horn:

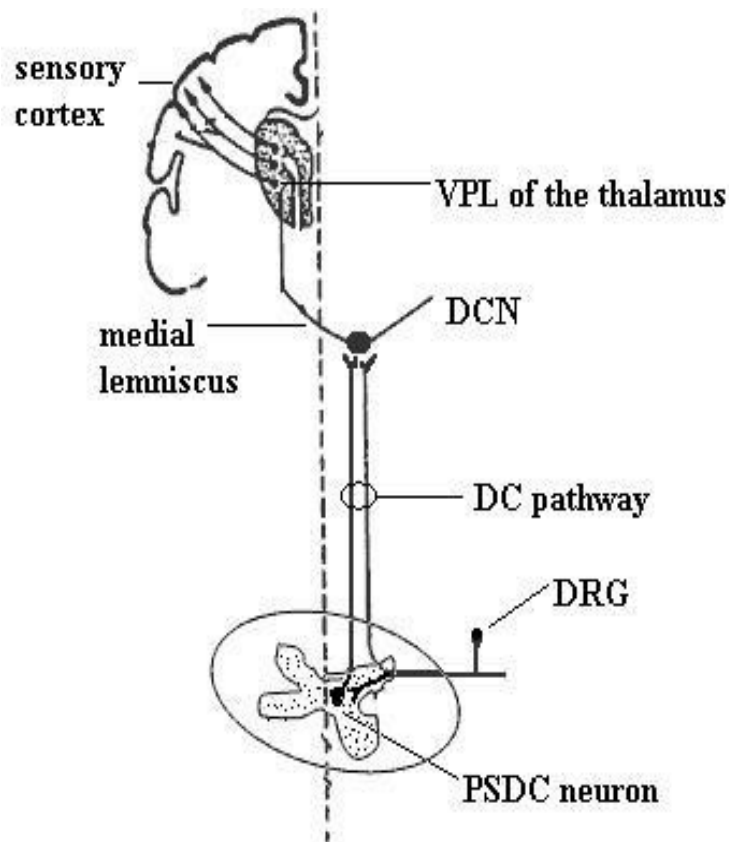
- *Lamina I*: specific nociceptor neurons
- *Lamina V*: multimodal (non-specific) neurons, which receive many convergent inputs, including visceral afferents.

There are several projection pathways from the spinal cord to the brain:

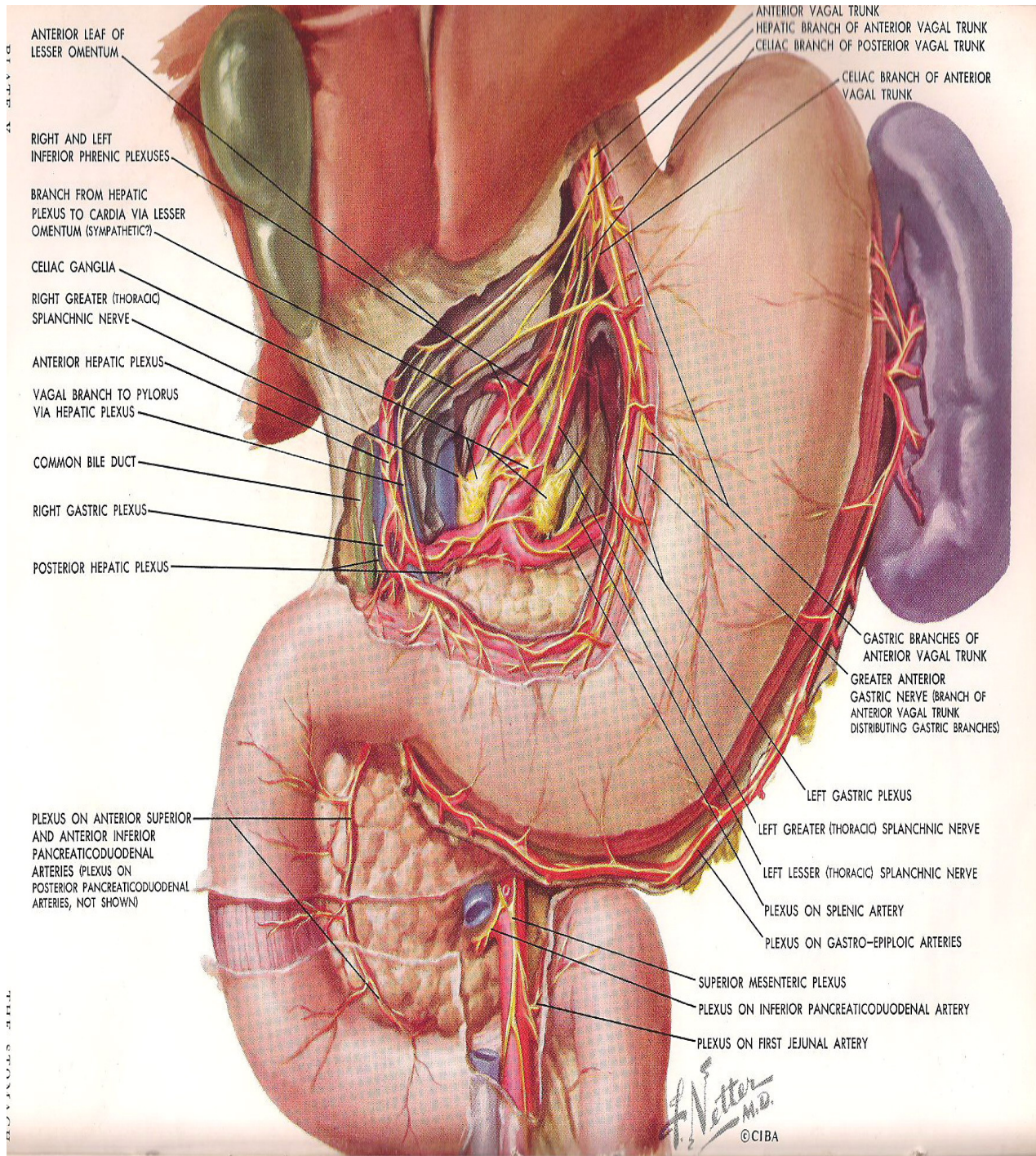
(a) A 'specific' pathway, probably the spinothalamic tracts, which projects to the somatosensory cortex. This pathway probably serves the 'sensory' and 'discriminative' aspects of pain.

(b) A 'non-specific' pathway, probably involving the spinoreticulothalamic tracts, which makes widespread and diffuse connections with many areas of the forebrain, including the limbic system. This system may serve the affective (or 'nasty') aspects of pain.

## VISCERAL PAIN PATHWAY



# INNERVATION OF STOMACH



## **POSTOPERATIVE PAIN**

- Pain is defined as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage.
- It is a complex process influenced by both physiological and psychological factors

## **EFFECTS OF POSTOPERATIVE PAIN**

- Postoperative pain can affects all organ systems and includes
- Respiratory - reduced cough, atelectasis, sputum retention and hypoxaemia
- Cardiovascular - increased myocardial oxygen consumption and ischaemia
- Gastrointestinal - decreased gastric emptying, reduced gut motility and constipation
- Genitourinary - urinary retention
- Neuroendocrine - hyperglycaemia, protein catabolism and sodium retention
- Musculoskeletal - reduced mobility, pressure sores and increased risk of DVT
- Psychological - anxiety and fatigue

## PHARMACOLOGICAL METHODS OF PAIN RELIEF

### SIMPLE ANALGESIA

- Paracetamol is a weak anti-inflammatory agent
- Modulates prostaglandin production in the central nervous system
- Can be administered orally or rectally or intramuscularly
- Best taken on a regular rather than 'as required' basis.
- Overdose results in hepatic necrosis
- Often combined with weak opiates (e.g. dihydrocodeine = Co-dydramol)

### NON-STEROIDAL ANTI-INFLAMMATORY AGENTS

- Inhibit the enzyme cyclo-oxygenase
- Reduces prostaglandin, prostacyclin and thromboxane production
- Also have weak central analgesic effect
- Often used for their 'opiate sparing' effects
- Side effects include:
  - Gastric irritation and peptic ulceration
  - Precipitation of bronchospasm in asthmatics
  - Impairment of renal function
  - Platelet dysfunction and bleeding

## **OPIATES**

- Most commonly used drugs are diamorphine, morphine, pethidine and pentazocine
- Diamorphine is a prodrug rapidly hydrolysed to morphine and 6-monoacetyl-morphine
- All act on mu receptors in brain and spinal cord
- Mu 1 receptors are responsible for analgesia

## **ROUTES OF OPIATE ADMINISTRATION**

- Oral - available for codeine, dihydrocodeine and oramorph
- Subcutaneous - useful for chronic pain relief
- Intramuscular - produces peaks and troughs in pain relief
- Intravenous - reliable but can produce sedation and respiratory depression
- Patient-controlled analgesia (PCA) - patient determines own analgesic requirement
  - 'Lock-out' period prevents accidental overdose
  - Safe as sedation occurs before respiratory depression
- Epidural or spinal

- Lipid soluble opiates (e.g. fentanyl) are normally used
- Produces good analgesia with reduced risk of side effects

## **LOCAL ANAESTHETIC AGENTS AND TECHNIQUES**

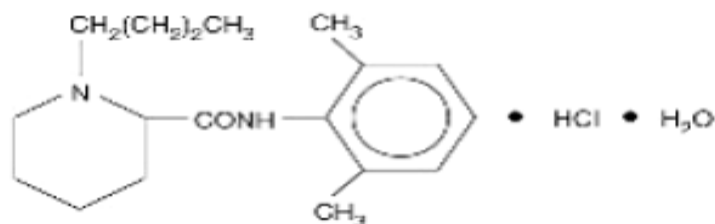
- Can be used by:
  - Wound infiltration
  - Nerve or nerve plexus blockade
  - Epidural infiltration
  - Intrathecal (spinal) administration
  - Intra peritoneal instillation as evidenced by recent trials
- Lignocaine has rapid onset but short duration of action
- Bupivacaine has more prolonged onset but shorter duration of action

## **BUPIVACAINE**

### **Chemical Structure**

Bupivacaine HCl is chemically designated as 2-piperidinecarboxamide, 1-butyl-N-(2,6-dimethylphenyl)-, monohydrochloride, monohydrate.





Bupivacaine chemically and pharmacologically to the aminoacyl local anesthetics.

- It is a homologue of mepivacaine and is chemically related to lidocaine.
- All three of these anesthetics contain an amide linkage between the aromatic nucleus and the amino, or piperidine group. They differ in this respect from the procaine-type local anesthetics, which have an ester linkage.

## Pharmacological Actions

Local anesthetics block the generation and the conduction of nerve impulses, presumably by increasing the threshold for electrical excitation in the nerve, by slowing the propagation of the nerve impulse, and by reducing the rate of rise of the action potential. In general, the progression of anaesthesia is related to the diameter, myelination, and conduction velocity of affected nerve fibers. Clinically, the order of loss



of nerve function is as follows: (1) pain, (2) temperature, (3) touch, (4) proprioception, and (5) skeletal muscle tone.

Systemic absorption of local anesthetics produces effects on the cardiovascular and central nervous systems (CNS). At blood concentrations achieved with normal therapeutic doses, changes in cardiac conduction, excitability, refractoriness, contractility, and peripheral vascular resistance are minimal.

### **Pharmacokinetics:**

The onset of action with Bupivacaine Hydrochloride is rapid and anaesthesia is long lasting. The duration of anaesthesia is significantly longer with Bupivacaine Hydrochloride than with any other commonly used local anaesthetic. It has also been noted that there is a period of analgesia that persists after the return of sensation. The pKa of bupivacaine (8.1) is similar to that of lidocaine (7.86). However, bupivacaine possesses a greater degree of lipid solubility and is protein bound to a greater extent than lidocaine.

Amide-type local anesthetics such as Bupivacaine Hydrochloride are metabolized primarily in the liver via conjugation with glucuronic acid. Patients with hepatic disease, especially those with severe hepatic disease, may be more susceptible to the potential toxicities of the amide-type local anesthetics. Pipecoloxylidine is the major metabolite of Bupivacaine Hydrochloride

Various pharmacokinetic parameters of the local anesthetics can be significantly altered by the presence of hepatic or renal disease, addition of epinephrine, factors affecting urinary pH, renal blood flow, the route of drug administration, and the age of the patient.

The half-life of Bupivacaine Hydrochloride in adults is 2.7 hours and in neonates 8.1 hours. The kidney is the main excretory organ for most local anesthetics and their metabolites. Urinary excretion is affected by urinary perfusion and factors affecting urinary pH. Only 6% of bupivacaine is excreted unchanged in the urine.

## Dosage

Maximum dosage: 2 mg/kg.

Maximum in 24 hours is 400 mg

## ADVERSE REACTIONS

**Central Nervous System Reactions:** These are characterized by excitation and/or depression. Restlessness, anxiety, dizziness, tinnitus, blurred vision, or tremors may occur, possibly proceeding to convulsions. However, excitement may be transient or absent, with depression being the first manifestation of an adverse reaction. This may quickly be followed by drowsiness merging into unconsciousness and respiratory arrest. Other central nervous system effects may be nausea, vomiting, chills, and constriction of the pupils.

**Cardiovascular System Reactions:** High doses or unintentional intravascular injection may lead to high plasma levels and related depression of the myocardium, decreased cardiac output, heartblock, hypotension, bradycardia, ventricular arrhythmias, including ventricular tachycardia and ventricular fibrillation, and cardiac arrest.

Patients over 65 years, particularly those with hypertension, may be at increased risk for developing hypotension while undergoing anesthesia with Bupivacaine Hydrochloride

## Contraindications

Bupivacaine Hydrochloride is contraindicated in obstetrical paracervical block anaesthesia. Its use in this technique has resulted in fetal bradycardia and death.

Bupivacaine Hydrochloride is contraindicated in patients with a known hypersensitivity to it or to any local anesthetic agent of the amide-type or to other components of Bupivacaine Hydrochloride solutions.

## Clinically Significant Drug Interactions

The administration of local anesthetic solutions containing epinephrine or norepinephrine to patients receiving monoamine oxidase inhibitors or tricyclic antidepressants may produce severe, prolonged hypertension. Concurrent use of these agents should generally be avoided. In situations when concurrent therapy is necessary, careful patient monitoring is essential.

## **ADRENALINE**

- Adrenaline can delay absorption and prolong duration of action
- Should not be used at sites of end-arteries (e.g. ear, fingers, penis)
- Act by reducing transmission along nerve fibres
- Work by blocking sodium channels in the nerve fibres
- Block pain-fibres first but can also result in
  - Neuromuscular blockade
  - Hypotension due to sympathetic blockade

## **ASSESSMENT OF PAIN**

- Pain is a subjective experience
- Observer assessment of patient behaviour is unreliable
- Pain should be assessed and recorded by:
  - Visual analogue scales
  - Verbal numerical reporting scale
  - Categorical rating scale

## **VERBAL RATING SCALES**

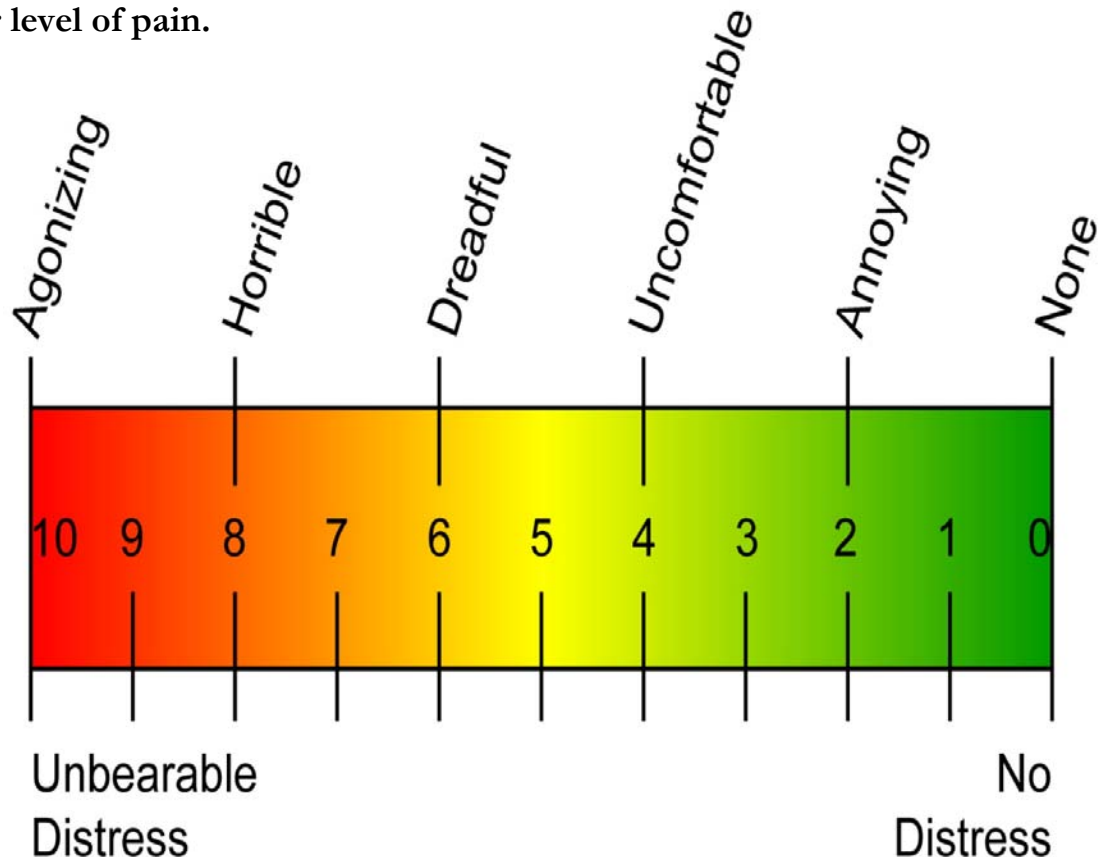
Verbal pain scales, as the name suggests, use words to describe pain. Words such as no pain, mild pain, moderate pain, and severe pain are used to describe pain levels. A score from 0 to 3 is assigned to each of those word pairs and is used to measure the pain level.

## NUMERICAL RATING SCALES

A numerical scale with the range of 0 to 10 is another type of pain scale that is used. The words "no pain" appear by the "0" and "worst pain possible" is found by the "10." You are asked to choose a number from 0 to 10 that best reflects your level of pain.

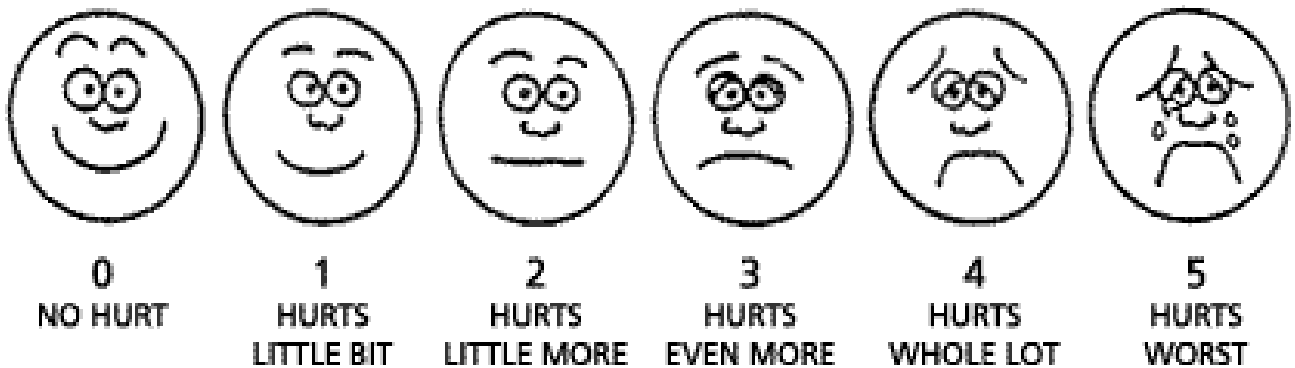
## VISUAL ANALOGUE SCALES

VAS or visual analogue scales use a vertical or horizontal line with words that convey "no pain" at one end and "worst pain" at the opposite end. You are asked to place a mark along the line that indicates your level of pain.



## WONG-BAKER FACES PAIN RATING SCALE

With the Wong-Baker pain scale, six faces are used that are numbered 0 to 5



Face 0 is a happy face (no hurt)

Face 1 is still smiling (hurts a little bit)

Face 2 is not smiling or frowning (hurts a little more)

Face 3 is starting to frown (hurts even more)

Face 4 is definitely frowning (hurts a whole lot)

Face 5 is crying although you don't have to cry to choose this face (hurts the worst)

The FACE pain scale would be particularly useful for children who may not have verbal skills to express their pain level.

## **BOTTOM LINE ABOUT PAIN SCALES**

The aforementioned pain scales focus on the severity of pain but they really don't delve into any other aspect of pain such as qualities of pain (sharp, dull, throbbing) or other characteristics of pain (annoying or unbearable). There are questionnaires designed for that purpose. The questionnaires gather more details about your pain than what can be derived from pain scale

## **FLACC SCALE**

FLACC stands for face, legs, activity, crying and consolability. It is an observer rated pain scale. The FLACC pain scale was designed for children between the ages of 2 and 7. However, some practitioners in adult settings may use the FLACC pain scale for people who are unable to communicate their pain. FLACC provides a pain assessment scale between 0 and 10.



<b>DATE/TIME</b>						
<b>Face</b> <b>0-No particular expression or smile</b> <b>1-Occasional grimace or frown, withdrawn, disinterested</b> <b>2-Frequent to constant quivering chin, clenched jaw</b>						
<b>Legs</b> <b>0 – Normal position or relaxed</b> <b>1 – Uneasy, restless, tense</b> <b>2 – Kicking, or legs drawn up</b>						
<b>Activity</b> <b>0 – Lying quietly, normal position, moves easily</b> <b>1 – Squirming, shifting back and forth, tense</b> <b>2 – arched, rigid or jerking</b>						
<b>Cry</b> <b>0 – No cry (awake or asleep)</b> <b>1 – Moans or whimpers: occasional complaints</b> <b>2 – Crying steadily, screams or sobs, frequent complaints</b>						
<b>Consolability</b> <b>0 – Content, relaxed</b> <b>1 – Reassured by occasional touching, hugging or being talked to, distractible</b> <b>2 – Difficult to console or comfort</b>						

**TOTAL SCORE**

## **CRIES SCALE**

The CRIES pain scale is an observer-rated pain assessment tool which is performed by a healthcare practitioner such as a nurse or physician. CRIES assesses crying, oxygenation, vital signs, facial expression and sleeplessness. The CRIES Pain Scale is generally used for infants 6 months old and younger.

## **THE PNEUMOPERITONEUM**

The laparoscopic surgeon should check the position of the patient prior to initiating the pneumoperitoneum. Positioning the patient on the operating room table is critical and will significantly increase intra-abdominal visualization.

Veress needles come in two lengths (120 cm and 150 cm); the longer version is obviously for obese patients.

### **1. PREPARATION OF THE ABDOMEN:**

The entire, anterior abdominal wall should be prepped from mid chest to mid thigh and as lateral as possible. Laparoscopic procedures can occasionally become very difficult and may require the insertion of additional trocars away from the original operating site.

## 2. GROUNDING THE PATIENT:

All patients, without exception, should be properly grounded.

## 3. INSERTION OF THE VERESS NEEDLE:

The safest access into the intra-abdominal cavity with a Veress needle is the umbilical area. The anterior abdominal wall is the thinnest at this level and all fascial layers are fused into single fascial planes. Thus, the operator should always attempt to insert the Veress needle at this site in the virgin abdomen.

In morbidly obese patients, use two traumatic towel clips or Lane's forceps on each side of the umbilicus to elevate the abdominal wall prior to attempt to insert the Veress Needle.

### *Step 1: Elevating the Anterior Abdominal Wall*

The anterior abdominal wall needs to be elevated in order to distance it from the intra-abdominal contents. This is done by grabbing the abdominal wall directly under the umbilicus with one hand. If the patient is overweight, two towel clamps can be used on each side of the umbilicus to achieve the same result.

### *Step 2: The Incision*

A 1 mm incision is made with a # 11 Scalpel at the umbilicus.

### *Step 3: Inserting the Veress Needle*

The spring function or the retraction capability of the Veress Needle is checked. The operating table should be in neutral or flat position. The needle is then slowly inserted into the incision. It is angled toward the pelvis and advanced. The operator should feel or sense the needle passing through two distinct planes.

The needle is advanced and withdrawn several times. If this is done easily and without obstruction, the tip is in proper position.

### *Step 4: The Saline Test*

Ten cc of normal saline is injected. This should be done easily. The abdominal pull is then released. The Veress needle is then filled to the rim with normal saline (or a open syringe can be used). The tension on the skin is resumed and the level of saline should immediately drop if the needle is in proper intra-abdominal position.



### *Step 5: Initiating the Insufflation*

The Veress needle is then connected to the CO<sub>2</sub> insufflation tubing (a filter should be used). Insufflation is initiated at a low flow. Intra-abdominal pressure recorded at this point should not exceed 8 mm Hg. Entry pressure at low flow should be checked immediately while the abdominal wall is still elevated. If higher, move the needle around or resume the pull on the skin or anterior abdominal wall. If the pressure is too high, the Veress needle is not in the right position and needs to be removed. Begin again.

If in place, switch to high flow and inflate the intra-abdominal cavity.

## USSC VERSASTEP

*The versa – step system is an integrated system combining a Nylon stretchable sheath over a Disposable Veress needle. Once inserted, the sheath is dilated by inserting the trocar [with a dilator in place]. The real advantage of this system is that it has no cutting entry blade, thus dramatically decreasing trocar site bleed and the potential for an intra abdominal injury. In addition, it creates a smaller fascial defect which does not need to be closed [up to 12 mm]*

### *Insertion of the VersaStep System Trocar*

Following insufflation the expandable needle system is inserted, the needle is withdrawn leaving the expandable sleeve in place.



A tapered blunt dilator is inserted through the sleeve, dilating the tract created by the needle.



The trocar is maintained in place by the expandable sleeve.



The blunt trocar is used to safely create a Pneumoperitoneum in the scarred abdomen. It is inserted by making an initial skin and a fascial incision. The fascial incision should be 1 to 1.5 cm in size. A long suture (2.0) is placed on each fascial edges. With finger dissection a tunnel or an opening into the intraabdominal cavity is gently created. The BluntPort\* is then inserted. The foamgrip anchoring device is set

and secured with the previously placed suture. The insufflation port is connected to the insufflation tubing and the pneumoperitoneum created.

IA 1 cm skin incision is made with a plain scalp. A telescope is inserted into the VISIPOINT OPTICAL TROCAR and the path of entry of the VISIPOINT OPTICAL TROCAR into intra-abdominal cavity is visualized. The VISIPOINT OPTICAL TROCAR is advanced slowly through the different planes of the abdominal wall. These planes are cut slowly with the blade of the VISIPOINT OPTICAL TROCAR (at the tip of the instrument) until the intraabdominal cavity is reached. Pneumoperitoneum must be created or abdominal wall elevation must be performed prior to the insertion of the VISIPOINT OPTICAL TROCAR

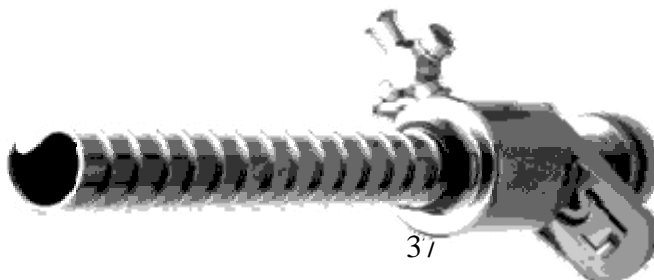




The Termanian Type trocar has greatly improved the safety and function of the re-usable trocar. This is the only re-usable trocar we use. It is inserted via a small incision without a pneumoperitoneum and rotating while advancing it. All the abdominal wall layers are well seen and visualized.



A laparoscopy can be performed without significant, deleterious effect with intraabdominal pressures up to 20 mm Hg. However, some laparoscopic inguinal hernia repairs require higher pressures in the 18 and 20 mm Hg. range to achieve necessary additional exposure.



## **GENERAL GUIDELINES ARE:**

The best operating intra-abdominal pressures are between 10 to 15 mm Hg. The visualization of this type of intra-abdominal pressure can be further enhanced by modifying the patients' position (operating table position to Trendelenburg to reverse, etc.)

Higher pressures in the 15 to 20 mm Hg range are suboptimal. There is a definite correlation with increased postoperative patient discomfort and recovery and the use of increased intra-abdominal pressure.

Pressures beyond 20 mm Hg are classified as dangerous with potential hemodynamic and pulmonary compromise and long term effects on the intra-abdominal wall musculature. When such pressures are used, some patients will actually report increased abdominal girth and a bloating post-operative feeling which persisted for months after the procedure in spite of intensive exercise.

## MATERIALS AND METHODS

### *Study Design:*

Prospective, Randomized, Double – Blind, Placebo – Controlled Study.

### *Sample Size and Randomization*

The study included 120 patients with uncomplicated, symptomatic cholelithiasis, subacute appendicitis admitted in department of general surgery, Coimbatore medical college hospital, Coimbatore between July 2007 to sep 2009.

All the investigated patients were managed by same team of surgeons. The study was approved by the Bioethics Committee of the Tamilnadu Dr.M.G.R.Medical University.

The randomization was based on each patient receiving a sealed envelope containing a random number selected from the table assigning the given individual to one of four groups equal in size ( $n = 30$ ).

- Group A received 2 mg/kg of bupivacaine diluted in 60 ml of normal saline immediately after the creation of pneumoperitoneum.
- Group B was administered 2 mg/kg of bupivacaine diluted in 60 ml of normal saline at the end of planned surgical procedure.
- Group C received 60 ml of normal saline immediately after the creation of pneumoperitoneum.
- Group D was given 60 ml of normal saline at the end of planned surgical after the creation of pneumoperitoneum.

## INCLUSION CRITERIA

- Uncomplicated, Symptomatic Cholelithiasis
- Subacute appendicitis
- Paltamos ligation
- Fundoplication
- Incisional hernia repair
- Age more than 12 years and less than 65 years
- ASA grade I and II

## **EXCLUSION CRITERIA**

- ASA grade III and more
- Patients with history of drug allergy
- Age less than 12 years and more than 65 years
- Pregnancy and lactation
- Previous extensive abdominal surgery
- Conversion to open surgery
- Prolonged administration of nonsteroidal antiinflammatory agents (NSAIDs) or other analgesics.

## **ANAESTHETIC PROTOCOL**

The two anesthesiologists involved in the study followed a strict protocol. All the individuals were premedicated with intravenous pethidine, midazolam, and paracetamol. Anesthesia was induced using fentanyl, thiopental, and pancuronium at the body mass–dependent dose. After endotracheal intubation, all the patients were provided with mechanical ventilation (isoflurane and oxygen mixture) and monitored by a capnograph to maintain the carbon dioxide (CO<sub>2</sub>) level in the expired air within the range of 4.0% to 4.5% throughout the procedure. In the course of the operation, the patients received 15 ml/ kg of

Ringer's solution in an intravenous infusion. In each case, a gastric tube was inserted for the duration of the procedure and removed before its termination. To prevent postoperative vomiting, intravenous metoclopramide was administered to each patient before awakening.

## SURGICAL TECHNIQUE

The procedures were performed by the same team of surgeons of surgical unit III involved in the study

The standard French four-port surgical technique was used for laparoscopic cholecystectomy. Four ports used for laparoscopic appendicectomy Local wound infiltration with 3 to 5 ml of 0.25% bupivacaine was routinely administered to all the patients before skin incisions. Access to the peritoneal cavity was established using veress needle.



The first trocar was placed through the umbilicus; and pneumoperitoneum with CO<sub>2</sub> was created. After introducing two trocars the patient was placed in the Trendelenburg position,

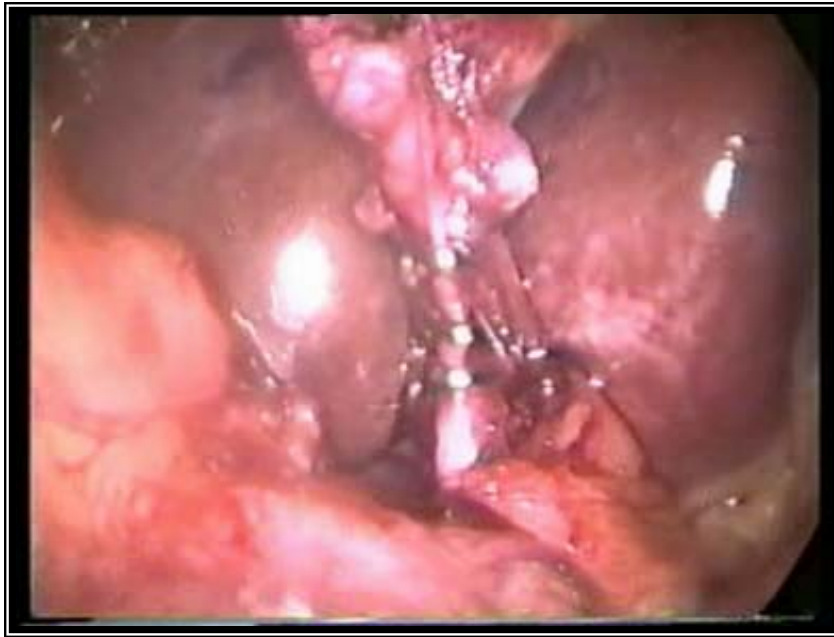
The standard suction-irrigation device with the attached 100-ml syringe was introduced through the second port cannula directed toward the area of dissection.

In groups A & C the solution was instilled immediately after the creation of pneumoperitoneum.

In groups B (bupivacaine) and D (saline), the solution instilled at the end of planned surgical procedure before removing the trocars.

An insufflation pressure of 12 mmHg was routinely used throughout the operations. The maximum flow of CO<sub>2</sub> administered at ambient temperature was electronically restricted to 2 l/min, both in the course of pneumoperitoneum creation and during further stages of the procedure.

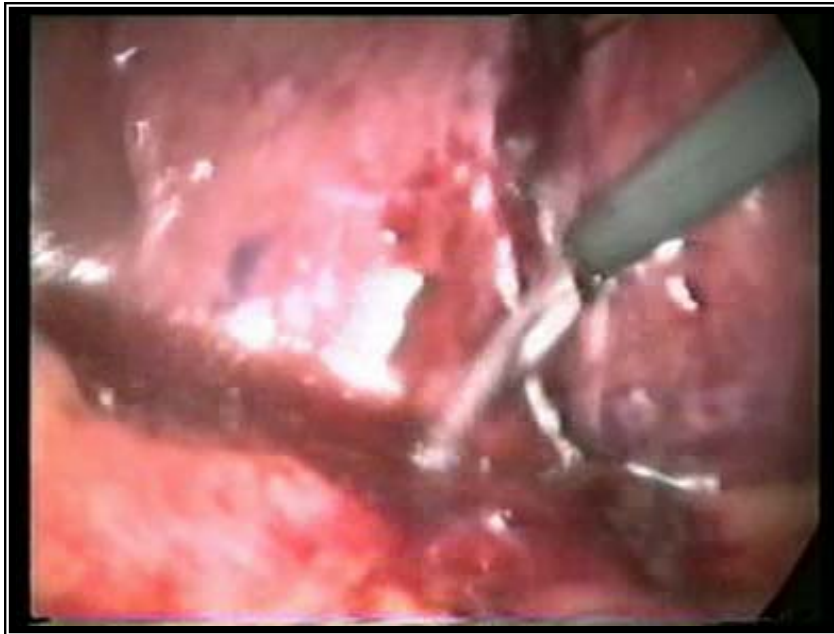
## LAPAROSCOPIC CHOLECYSTECTOMY





## **LAPAROSCOPIC CHOLECYSTECTOMY**

### **INTRA PERITONEAL BUPIVACAINE INSTILLATION**



## **LAPAROSCOPIC APPENDICECTOMY**



## POST OPERATIVE PAIN ASSESSMENT

All data were entered into a dedicated proforma. A detailed statistical analysis included sex, age, body mass index (BMI), ASA grade, medical history before operation, duration of surgery, postoperative pain assessed by a visual analog scale (VAS), the time lapse between the operation and the first demand for analgesics by the patient, the total daily analgesia requested throughout the initial 48 h after the surgery, and the incidence and intensity of shoulder tip pain.

The primary end points were postoperative pain intensity on the VAS 4, 8, 12, 24, and 48 h after LC and the incidence of shoulder tip pain. The secondary end points included the latency of nurse-controlled analgesia (NCA) activation, the analgesia request rate, and analgesic consumption. The number of patients who needed treatment was estimated according to the principle of detecting a 20% difference in pain intensity with a 90% probability at  $p$  assumed to be less than 0.05.

The statistical analysis was based on chisquare and Student t-tests. The intensity of postoperative pain was assessed with the patient at rest using VAS, with evaluation at 4, 8, 12, 24, and 48 h postoperatively.

Neither the patients nor the nurses knew the relevant group assignment. Postoperative pain scores were obtained by an independent clinical investigator, who was also blinded. The patients were aware that the scale served to analyze the intensity of pain alone, including shoulder tip pain, if present, and was not a representation of their generalized postoperative discomfort.

For further analysis, the data were treated as parametric. The nursing team recorded episodes of vomiting and nausea. All the patients received elective intra muscular diclofenac analgesia administered on demand (NCA), If required, NCA was continued with oral administration 12 h postoperatively. All postoperative analgesic requirements, including the latency of the first NCA activation, pain scores, and relevant clinical parameters, were recorded in a dedicated proforma.

The patients were allowed to assume erect position, mobilized, and given an oral diet 24 h after the surgery. An overnight hospital stay was mandatory for all the patients. All the subjects were seen by the surgeons involved in the study during follow-up visits at the outpatient surgical department 3 weeks after the operation.

## OBSERVATIONS AND RESULTS

Between Jan 2008 to July 2009 Patients were admitted to our surgical department at Coimbatore Medical College Hospital for the Management of uncomplicated Cholelithiasis, sub acute appendicitis, fundoplication, Palamos ligation, Incisional hernia repair. In all, 120 patients were randomized to four groups equal in size ( $n = 30$ ). There were no significant differences between the groups with respect to age, sex, BMI, ASA grade, and mean duration and intensity of cholelithiasis-associated complaints (Table 1). Patients who had laparotomy because of difficulty and complications were excluded from the study. The mean operating time was comparable between the groups.

No major intraoperative complications such as bleeding appeared in any of the patients. Gallbladder perforation and resultant bile spillage into the subhepatic space occurred in the course of coagulation hook cholecystectomy for 1 patient in group A, no patients in group B, 2 patients in group C, and 1 patient in group D, and was followed by a thorough suctioning and rinsing with a small volume of saline. The differences between the groups were not significant.

The mean intensity of postoperative total abdominal pain as assessed by VAS scale was significantly lower in group A than in group B patients, by mean values of 20.3% and 15.5% at 4 and 8 h after the operation, respectively. This difference became nonsignificant at 12 h postoperatively. Pain relief remained significantly higher in group A than in group C patients throughout the entire 48-h follow-up period, whereas in group C versus group D patients, it was limited to a 24-h period only, being nonsignificant at 48 h postoperatively.

None of group A patients reported shoulder tip pain, whereas in groups B, C, and D, it was reported, respectively, by 3, 6, and 7 individuals at 4 h, by 2, 4, and 4 individuals at 8 h, and by 0, 3, and 4 individuals at 12 h after the operation. This difference was found to be significant for group A versus group C at 4 and 8 h after the operation ( $p$  value = 0.01 and  $p$  value = 0.03, respectively), whereas borderline significance was observed for group A versus group B at 4 h and for group A versus group C at 12 h postoperatively ( $p$  value = 0.07 in both cases).

The latency time from the end of the operation to the first activation of NCA after a patient's demand was significantly longer in group A than in group B patients ( $p$  value < 0.001). However, this

parameter was also significantly higher for the group B patients than for the control subjects. The analgesia request rate was significantly lower after the operation for group A than for group B (21 vs 27)

**Table 1. Characteristics of the patients**

No. of Patients Sex (F/M) Age (Years) BMI (Kg/M2) Operation Time (Min)

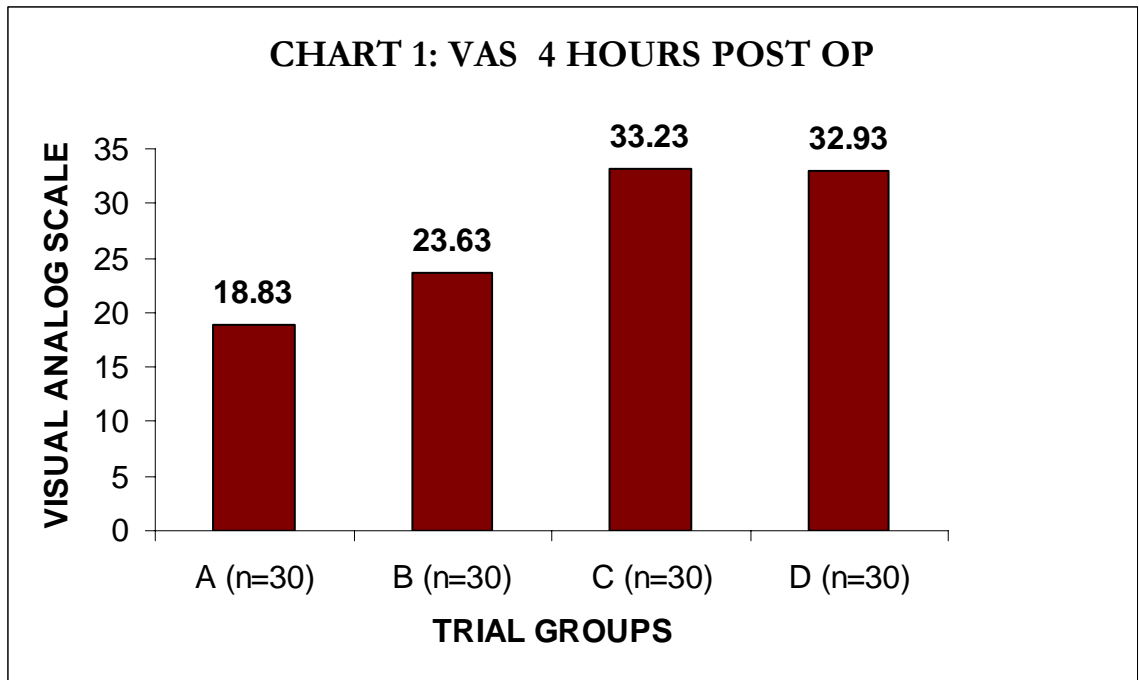
Group	No. patients	Sex m / f	Age years	BMI (Kg / M <sup>2</sup> )	Operation time (min)
A	30	15/15	35.26	27.3 ± 3.2	56.1 ± 12.7
B	30	17/13	33.76	27.1 ± 3.1	59.5 ± 11.7
C	30	14/16	33.00	26.9 ± 2.9	55.3 ± 12.7
D	30	11/19	34.66	27.6 ± 3.4	55.1 ± 10.6

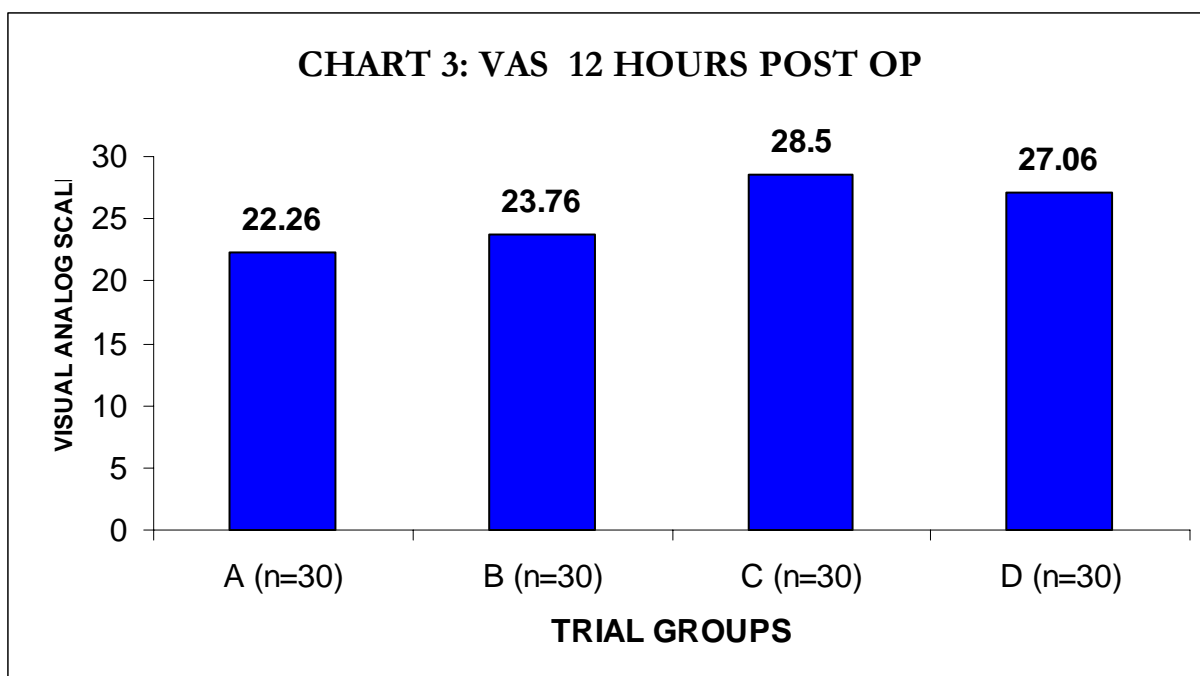
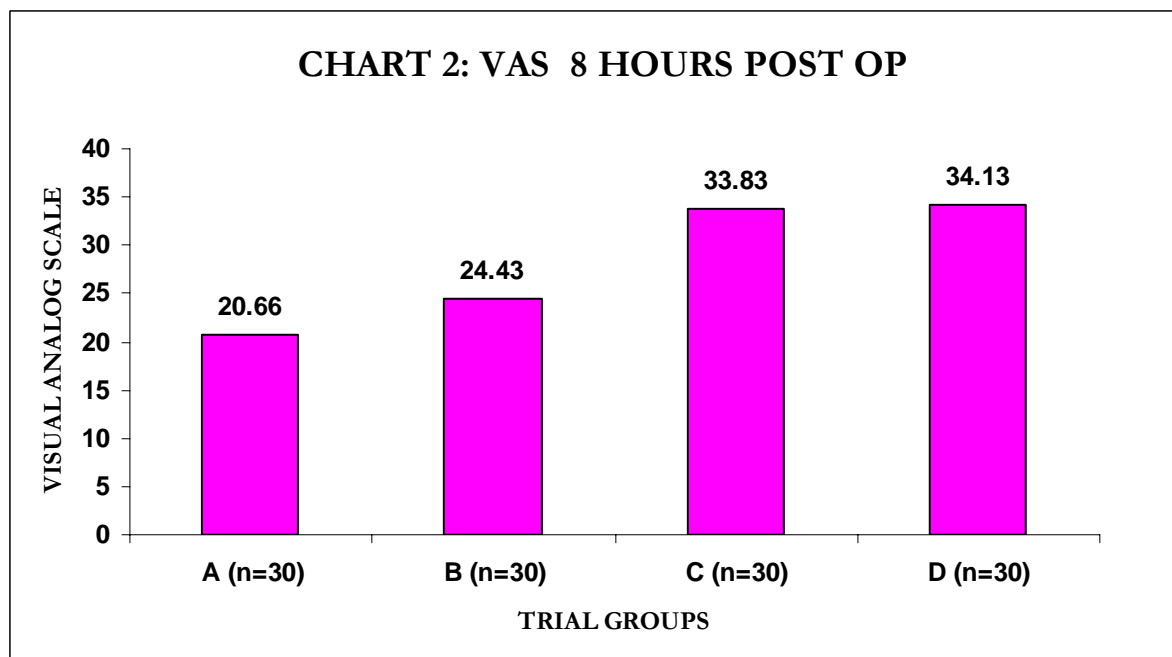
BMI, body mass index There were no significant differences between the groups

**Table 2. Postoperative Pain Score**

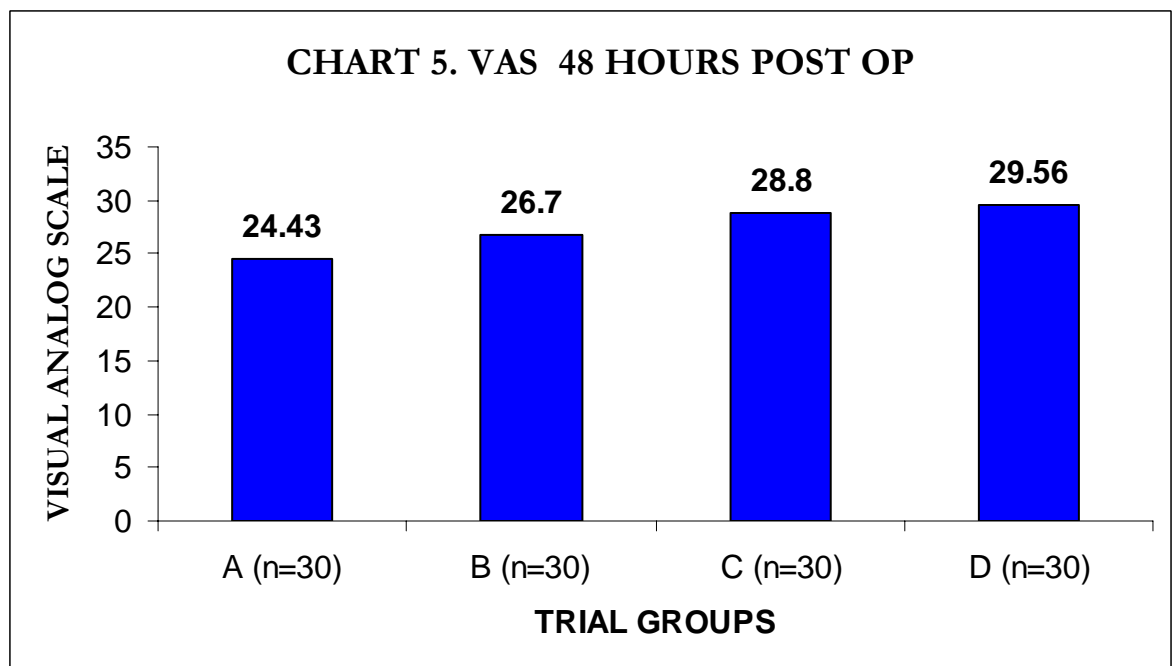
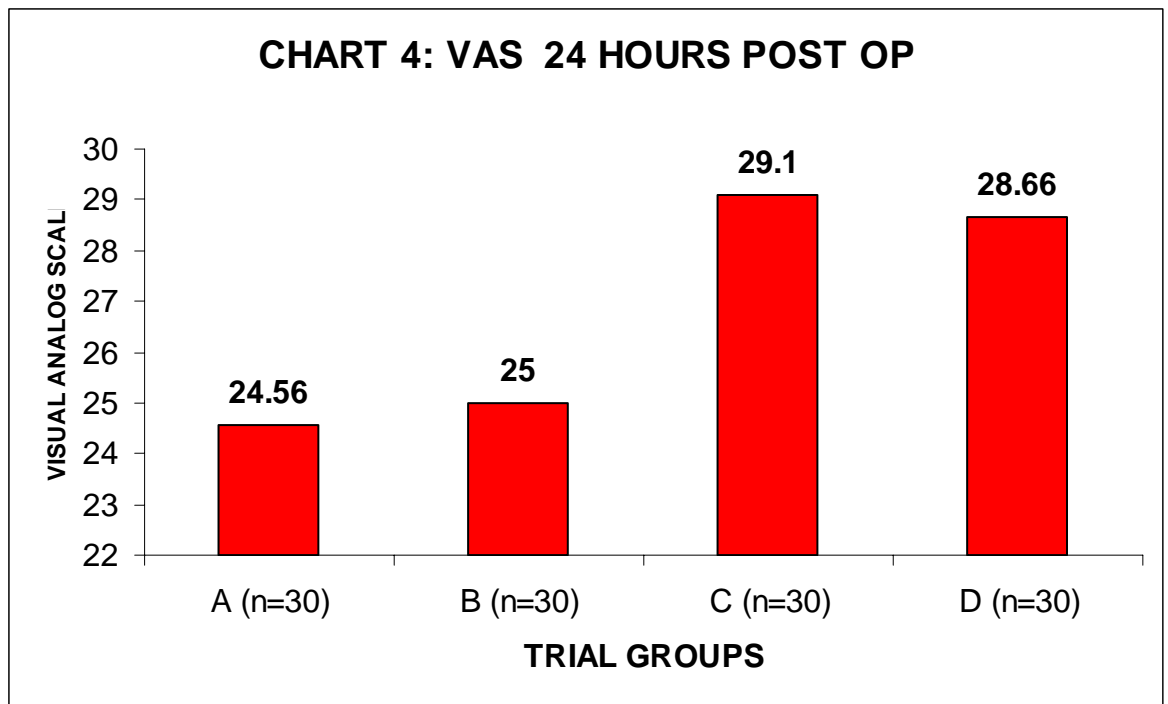
**Time After Operation (h) A (n = 30) B (n = 30) C (n = 30) D (n = 30)**

<b>Time of operation</b>	<b>A (n=30)</b>	<b>B (n=30)</b>	<b>C (n=30)</b>	<b>D (n=30)</b>
<b>4</b>	<b>18.83 ± 4.63</b>	<b>23.63 ± 5.63</b>	<b>33.23 ± 5.44</b>	<b>32.93 ± 4.49</b>
<b>8</b>	<b>20.66 ± 4.08</b>	<b>24.43 ± 5.27</b>	<b>33.83 ± 5.07</b>	<b>34.13 ± 4.42</b>
<b>12</b>	<b>22.26 ± 3.30</b>	<b>23.76 ± 4.86</b>	<b>28.50 ± 3.52</b>	<b>27.06 ± 3.80</b>
<b>24</b>	<b>24.56 ± 2.97</b>	<b>25.00 ± 3.82</b>	<b>29.10 ± 2.91</b>	<b>28.66 ± 3.63</b>
<b>48</b>	<b>24.43 ± 6.08</b>	<b>26.70 ± 3.68</b>	<b>28.80 ± 3.63</b>	<b>29.56 ± 4.07</b>

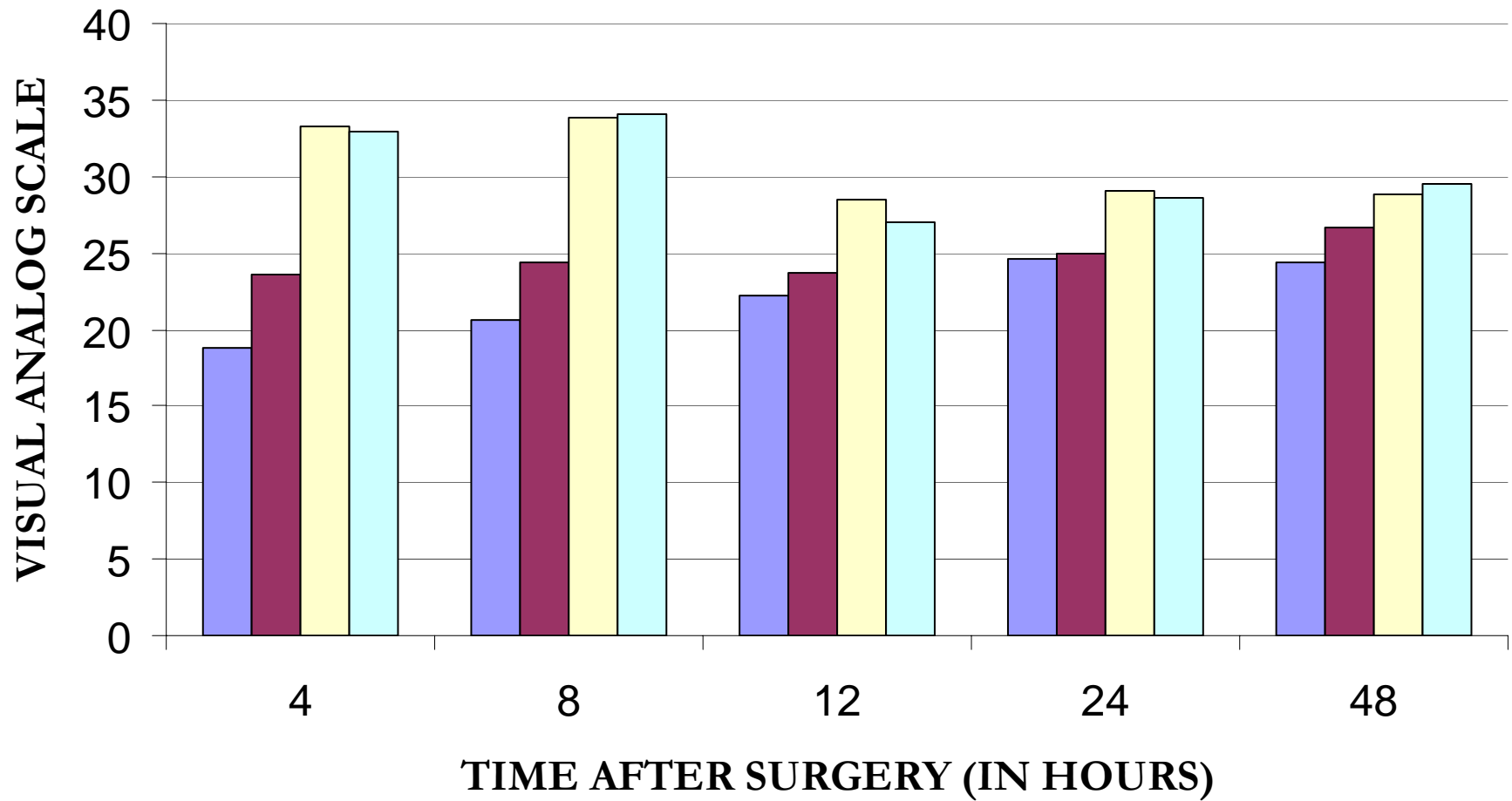








## VISUAL ANALOG SCORE COMPARISON



a Pain scores assessed by the visual analogue scale b None of the values for C vs D were significant c Student's t-test 1090 27 at 4 h and 20 vs 27 patients at 8 h, respectively). However, this difference became nonsignificant at 12 h postoperatively.

Group A had a significantly lower analgesia requirement rate throughout the entire 48-h period after the operation than group C, whereas the differences in the analgesia request rate between groups B and group D were nonsignificant throughout the entire 48-h follow-up period. The mean daily NSAID consumption was significantly lower in group A (150.00 mg) than in group B (212.50 mg) on day 1 postoperatively ( $p < 0.001$ ). However, on day 2, this difference became nonsignificant. Group A versus group C patients and group B versus D patients required significantly less analgesia.

**Table 3. Postoperative Pain Score**

**Time after operation p Value (A vs B) p Value (A vs C) p Value  
(B vs D)**

<b>Time of operation (HR)</b>	<b>P Value (A vs B)</b>	<b>P Value (A vs C)</b>	<b>P Value (B vs D)</b>
<b>4</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
<b>8</b>	<b>0.003</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
<b>12</b>	<b>0.16</b>	<b>&lt;0.001</b>	<b>0.007</b>
<b>24</b>	<b>0.62</b>	<b>&lt;0.001</b>	<b>0.01</b>
<b>48</b>	<b>0.09</b>	<b>0.03</b>	<b>0.19</b>

**Table 4: Latency of the first nurse-controlled analgesia request (LNCAR) (min), analgesia request (AR) (number of individuals who required pain medication), and analgesic consumption (AC) mean daily milligrams of diclofenac sodium.**

	Time after surgery	A (n = 30)	B (n = 30)	C (n = 30)	D (n = 30)
<b>LNCAR</b>		426.8 ± 57.2	307.0 ± 39.8	109.3 ± 51.0	109.0 ± 46.5
<b>AR</b>	4 h	21	27	28	29
<b>AR</b>	8 h	20	27	28	27
<b>AR</b>	12 h	12	16	20	21
<b>AR</b>	24 h	9	15	18	17
<b>AR</b>	48 h	7	11	15	16
<b>Ac</b>	Day 1	150.00	212.50	235.00	235.00
<b>Ac</b>	Day 2	17.5	27.5	37.5	40.00

**Table 5. Latency of the first nurse-controlled analgesia request (LNCAR) (min), analgesia request (AR) (number of individuals who required pain medication), and analgesic consumption (AC) mean daily milligrams of diclofenac sodium.**

	Time after surgery	p Value (A vs B)	p Value (A vs C)	p Value (B vs D)
<b>LNCAR</b>		<b>&lt;0.001b</b>	<b>&lt;0.001b</b>	<b>&lt;0.001b</b>
<b>AR</b>	<b>4 h</b>	<b>0.05</b>	<b>0.02</b>	<b>0.30</b>
<b>AR</b>	<b>8 h</b>	<b>0.03</b>	<b>0.01</b>	<b>1.0</b>
<b>AR</b>	<b>12 h</b>	<b>0.30</b>	<b>0.04</b>	<b>0.18</b>
<b>AR</b>	<b>24 h</b>	<b>0.11</b>	<b>0.02</b>	<b>0.60</b>
<b>AR</b>	<b>48 h</b>	<b>0.26</b>	<b>0.03</b>	<b>0.19</b>
<b>Ac</b>	<b>Day 1</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
<b>Ac</b>	<b>Day 2</b>	<b>0.23</b>	<b>0.002</b>	<b>0.004</b>

## DISCUSSION

Pain after laparoscopic surgery is considered to arise from three main sources: the incision sites within the abdominal wall, the pneumoperitoneum in association with both local (peritoneal and diaphragmatic stretching, ischemia, acidosis) and systemic (hypercarbia causing sympathetic nervous system excitation resulting in amplification of the local tissue inflammatory response) changes, and the postcholecystectomy wound within the liver (visceral pain).

Total abdominal pain after laparoscopic surgery covers all these aspects, with the largest component (50–70%) arising from the incision sites, followed by the pneumoperitoneum (20–30%) and cholecystectomy (10–20%) . Most laparoscopic operations are performed using a pneumoperitoneum with CO<sub>2</sub> insufflated into the abdominal cavity. Distension of the abdominal cavity by insufflated gas results in diaphragmatic stretching that involves shoulder tip pain. The exact mechanism of shoulder tip pain has yet to be clarified.

The proposed mechanisms include phrenic nerve neuropraxia of short duration, stretching of the subdiaphragmatic fibers by an increased

concavity of the diaphragm induced by pneumoperitoneum, loss of visceral surface tension after the creation of pneumoperitoneum leading to increased weight on the diaphragmatic attachments of the liver, and peritoneal damage caused by chemical irritation, ischemia, and compression. Because pain after laparoscopic surgery is multifactorial, a multimodal approach to postoperative pain management has been suggested.

Many different methods have been used successfully to diminish the intensity of pain after laparoscopic surgery, including a low-pressure pneumoperitoneum, a gasless technique for creating a working space, local wound anesthetic infiltration, saline washout , and instillation of the subdiaphragmatic region with a local anesthetic . The latter method, usually using bupivacaine, has been evaluated in more than a dozen trials. However, the results of the cited studies have been conflicting, with the majority demonstrating some benefit in terms of pain reduction, although the magnitude of this reduction and the duration of the effect have been limited. The following observations have been made.

The timing of the intraperitoneal administration of bupivacaine seems to be of great importance (before the dissection rather than at the end of laparoscopic surgery, as well as the volume of the solution and the



appropriate method of solution application, allowing for a good propagation of the fluid to both the left and right sides of the diaphragm and to the area of dissection to maximize pain reduction. In only a few trials was a local anesthetic administered intraperitoneally before dissection . Although these trials have demonstrated a benefit in terms of pain reduction, in none of them was a real preemptive analgesia used. The term “preemptive analgesia” means preoperative administration of drugs that modulate the development of the nociception process in the intra-and postoperative periods, which results in a reduced postoperative requirement for painkillers. Preemptive analgesia prevents the establishment of central sensitization to noxious stimuli by the decreased enhancement of pain sensation .The current trial was designed to evaluate preemptive analgesia with intraperitoneal instillation of bupivacaine before the potential nociceptive stimuli appearance.

The incidence of shoulder tip pain and the impact of pain intensity on the analgesia request rate and analgesia consumption were not assessed because all the patients were receiving equal doses of analgesics postoperatively. The main advantage of using local anesthetics is that they do not have the adverse effects of opioids, which may delay recovery and discharge from the hospital. They also are safe,

because the range of mean plasma concentration after intraperitoneal instillation of plain bupivacaine in the amount of 150 mg is well below the toxic concentration level.

The current study has proved that the application of peritoneal instillation with bupivacaine immediately after the creation of pneumoperitoneum is superior to its use at the end of planned surgical procedure the creation of pneumoperitoneum. Findings have demonstrated both the primary and secondary end points to be favorable for preemptive analgesia immediately after the creation of pneumoperitoneum. However, the total abdominal pain-diminishing effect has been rather short lived because significant differences have been observed only at 4 and 8 h postoperatively, whereas the reduction of shoulder tip pain to zero in the group receiving preemptive analgesia immediately after the creation of pneumoperitoneum has been a huge benefit previously unobtainable with any routinely used method.

The differences in total abdominal pain have become nonsignificant at 12 h postoperatively, probably because at that time the patients have been mobilized and the total abdominal pain has started to reflect the incision site pain instead of the diaphragm-related pain, the latter being more important in the early postoperative hours. This

observation is supported by a longer latency time from the end of the operation to the first activation of NCA demanded by the patients in group A than by the patients in group B, because during the immediate postoperative hours, the diaphragm-related respiratory pain constitutes the larger component of the total abdominal pain than the painful sensation arising from incision sites.

Moreover, a lower analgesia requirement rate in group A than in group B has been observed (a significant difference at 4 and 8 h postoperatively), as well as lower analgesic requirements during the initial 24 h after the surgery. These effects are probably secondary to a decreased central appreciation of pain and a lower level of pain expectancy, often leading to an increased analgesia request in anticipation of pain.

## CONCLUSION

Intraperitoneal instillation of bupivacaine in saline immediately after the creation of pneumoperitoneum has improved the surgical outcome after laparoscopic surgery by significantly diminishing abdominal pain, in addition to elimination of shoulder tip pain. It also decreases the post operative requirement of analgesic drugs.

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## **PROFORMA FOR POSTOPERATIVE PAIN EVALUATION**

NAME	:	AGE	:	SEX:
ADDRESS	:	WEIGHT	:	BMI:
I. P. NO	:	OCCUPATION:		
DOA	:	DOS	:	DOD:
H/O SMOKING	:			
H/O ALCOHOL INTAKE	:			
H/O ANY PSYCHIATRIC ILLNESS/ CHRONIC AILMENT	:			
H/O PREVIOUS DRUG INTAKE	:			
COMORBID CONDITIONS	:			
ANAESTHESIA TYPE	:	GA / EPIDURAL		
DURATION OF ANAESTHESIA	:			
DRUGS USED DURING ANAESTHESIA	:			
LAPAROSCOPIC PROCEDURE	:			
NO. OF PORTS	:			
GAS USED FOR PNEUMOPERITONEUM	:			
INTRA ABDOMINAL PRESSURE	:			
DURATION OF SURGERY	:			
INTRAPERITONEAL SOLUTION USED	:	BUPIVACAINE / NORMALSALINE		
TIME OF INSTILLATION	:			
POST OP ANALGESIA USED	:			

NAME :

AGE

:

SEX:

IP.No:

## ABDOMINAL PAIN

4 HRS		8 HRS		12 HRS		24 HRS		48 HRS	
PR		PR		PR		PR		PR	
BP		BP		BP		BP		BP	
RR		RR		RR		RR		RR	
100		100		100		100		100	
90		90		90		90		90	
80		80		80		80		80	
70		70		70		70		70	
60		60		60		60		60	
50		50		50		50		50	
40		40		40		40		40	
30		30		30		30		30	
20		20		20		20		20	
10		10		10		10		10	
0		0		0		0		0	

NAME :

AGE

:

SEX:

IP.No:

## SHOULDER PAIN

4 HRS	8 HRS	12 HRS	24 HRS	48 HRS
PR	PR	PR	PR	PR
BP	BP	BP	BP	BP
RR	RR	RR	RR	RR
100	100	100	100	100
90	90	90	90	90
80	80	80	80	80
70	70	70	70	70
60	60	60	60	60
50	50	50	50	50
40	40	40	40	40
30	30	30	30	30
20	20	20	20	20
10	10	10	10	10
0	0	0	0	0

### GROUP A

S.No	Name	Age/Sex	IP No	Ward	Date of Surgery	Diagnosis	Surgical Procedure
1	Anusiya devi	22/F	2384	FS3	21/01/2008	Intraabdominal lymphadenopathy	Laparoscopic LN Biopsy
2	Prema	35/F	10477	FS3	07/03/2008	Sub Acute Appendicitis	Lap. Appendicectomy
3	Sheela	27/F	5430	Special	04/02/2008	Incisional hernia	Lap. Hernia repair
4	Neela veni	42/F	5810	FS3	11/02/2008	Calculous Cholecystitis	Lap. Cholecystectomy
5	Papathi	55/F	3900	FS3	08/02/2008	Calculous Cholecystitis	Lap. Cholecystectomy
6	Saraswathi	52/F	10415	FS3	03/03/2008	Incisional hernia	Lap. Hernia repair
7	Rajamani	18/F	19588	FS3	21/04/2008	Sub Acute Appendicitis	Lap. Appendicectomy
8	Rena	29/F	43473	FS3	18/08/2008	Sub Acute Appendicitis	Lap. Appendicectomy
9	Muthulakshmi	30/F	50569	FS3	19/09/2008	Calculous Cholecystitis	Lap. Cholecystectomy
10	Revathi	28/F	44739	FS3	08/09/2008	Iliocaecal intussusception	Laparocopy & proceed
11	Muthulakshmi	21/F	57545	FS3	24/10/2008	Lap. Cholecystectomy	Lap. Appendicectomy
12	Pusba	40/F	52045	FS3	06/10/2008	Incisional hernia	Lap. Hernia repair
13	JOTHI MANI	30/F	3681	FS3	30/01/09	Sub Acute Appendicitis	Lap. Appendicectomy
14	RANI	30/F	72739	FS3	06/03/09	Appendicitis	Lap. Appendicectomy
15	Shanthi	52/F	17683	FS2	11/04/2009	Sub Acute Appendicitis	Lap appendicectomy

16	Mohankumar	25/M	26546	MS4/S 3	15/05/09	Sub Acute Appendicitis	Lap. appendicectomy
17	Sudhakar	28/M	67182	MS4/S 3	12/12/08	Recurrent hernia	Lap. TAPP
18	Kumar	38/M	60229	MS4	07/11/2009	Left inguinal hernia	Lap. TAPP
19	Nagamanika m	13/M	33913	MS5	18/06/2009	Sub Acute Appendicitis	Lap. appendicectomy
20	Lazar	49/M	43189	MS5	13/08/2009	Right bubunocoele	Lap. TAPP
21	Sentil Raja	25/M	27951	MS5	23/05/2009	Calculous Cholecystitis	Lap. Cholecystectomy
22	KJOSEPH	53M	50513	MS4	03/10/08	Calculous Cholecystitis	Lap. Cholecystectomy
23	Sunmugam	26/M	20291	MS4/S 3	17/04/09	Sub Acute Appendicitis	Lap. appendicectomy
24	Vijayan	59/M	18250	MS4	11/04/08	Recurrent inguinal hernia	Laparoscopic repair
25	GOPAL	50M	41994	MS4	01/09/08	Hiatus hernia	Lap fundoplication
26	VIJAYAN	33M	47919	Ms4	08/09/08	Rt. Varicocle	Dlagnostic laproscopy
27	Senthil	33/M	23540	MS5	29/04/2009	Right inguinal hernia	Lap. TAPP
28	Kuppusamy	55/M	50461	MS5	12/09/2009	Left inguinal hernia	Lap. TAPP
29	Ravichandran	45/M	61678	MS4	28/11/09	B/L inguinal hernia	Lap.inguinal
30	Govindaraj	15/M	65862	MS4/S 3	05/12/08	Congenital hernia	Lap. repair



### GROUP B

S.No	Name	Age/Sex	IP No	Ward	Date of Surgery	Diagnosis	Surgical Procedure
1	Arif Begam	35/F	3855	FS3	01/02/2008	Para Umbilical hernia	Lap. Hernia repair
2	Beena	26/F	6597	FS3	11/02/2008	Recurrent Appendicitis	Lap. Appendicectomy
3	Momtaz	24/F	19750	FS3	21/04/2008	Sub Acute Appendicitis	Lap. Appendicectomy
4	Saraswathi	40/F	36800	FS3	25/08/2008	Calculous Cholecystitis	Lap. Cholecystectomy
5	Rajathi	45/F	44869	FS3	29/08/2008	Sub Acute Appendicitis	Lap. Appendicectomy
6	Pusba	40/F	52045	FS3	06/10/2008	Incisional hernia	Lap. Hernia repair
7	mariyammal	50/F	55707	FS3	10/11/2009	Sub Actue intestinal obstruction	Laparocopy & proceed
8	Rajeswari	35/F	62340	FS3	12/01/09	Sub Acute Appendicitis	Lap. Appendicectomy
9	JAYA	27/F	8047	FS3	20/02/09	Calculous Cholecystitis	Lap. Cholecystectomy
10	Priya	22/F	24882	FS2	07/05/2009	Sub Acute Appendicitis	Lap. appendicectomy
11	Kalpana	13/F	29433	FS2	30/05/2009	Sub Acute Appendicitis	Lap. appendicectomy
12	Abiba	29/F	40470	FS2	23/07/2009	Sub Acute Appendicitis	Lap. appendicectomy
13	Devi	38/F	40582	FS2	08/08/2009	Sub Acute intestinal obstruction	Lap. adhesiolysis

14	Veeran	51/M	8005	MS4/S3	16/02/09	Calculus Cholecystitis	Lap. Cholecystectomy
15	YOGA RAJ	27/M	153	MS4	07/01/08	Sub Acute Appendicitis	Lap. Appendicectomy
16	Eswaran	59/M	10466	MS4	14/03/08	Ca sigmoid colon	Laparoscopy & proceed
17	SHAIK MOIDEEN	17/M	14330	MS4	24/03/08	Sub Acute Appendicitis	Lap. Appendicectomy
18	Ponnuraj	27/M	141608	MS4	14/04/08	Indirect hernia	Lap. Hernia repair
19	Venu prasanth	25/M	52044	MS4	26/09/08	Calculous Cholecystitis	Lap. Cholecystectomy
20	Anilkumar	25/M	30905	MS5	04/06/2009	Sub Acute Appendicitis	Lap. appendicectomy
21	Rangaswamy	39/M	33849	Ms5	23/07/2009	Incisional hernia	Lap. IPOM
22	Govindarayan	65/M	61699	MS4	21/11/08	B/L inguinal hernia	Lap. repair
23	Giripoosaran	30/M	65868	MS4/S3	05/12/08	Sub Acute Appendicitis	Lap. appendicectomy
24	RAMAN	21/M	64097	MS4/S3	01/12/08	Inguinal Hernia	Lap. Repair
25	Samy aya	49/M	61804	MS4	14/11/08	direct inguinal hernia	
26	Sentil Kumar	20/M	41810	MS5	13/08/2009	Calculus Cholecystitis	Lap. Cholecystectomy
27	Thangam	31/M	50607	MS5	19/09/2009	Left inguinal hernia	Lap. TAPP
28	Subramaniya m	55/M	23569	Special	28/05/2009	Secondaries Liver	Diagnostic Laparoscopy
29	Paneerselvam	35/M	26408	MS5	16/05/2009	Right recurrent inguinal hernia	Lap. TAPP
30	GOKUL	13M	50568	MS4	19/09/08	Rt sided Indirect hernia	Lap. Hernia repair

### GROUP C

S.No	Name	Age/Sex	IP No	Ward	Date of Surgery	Diagnosis	Surgical Procedure
1	Rubeena	20/F	49153	FS3	15/09/2008	Sub Acute Appendicitis	Lap. Appendicectomy
2	sarojini	33/F	70081	FS3	02/01/09	Sub Acute Appendicitis	Lap. Appendicectomy
3	lakshmi	48/F	42107	FS3	18/08/2008	G.B.polyp	Lap. Cholecystectomy
4	Lakshmi	27/F	47724	FS3	15/09/2008	Calculous Cholecystitis	Lap. Cholecystectomy
5	SHANTHI	29/F	3591	FS3	23/01/09	Calculous Cholecystitis	Lap. Cholecystectomy
6	Shanthi	43/F	13683	FS2	09/04/2009	Sub Acute Appendicitis	Lap. appendicectomy
7	Renuka Devi	19/F	24921	FS2	09/05/2009	Sub Acute Appendicitis	Lap. appendicectomy
8	Mariyamal	50/F	26451	FS2	16/05/2009	Calculous Cholecystitis	Lap. Cholecystectomy
9	Amutha vali	13/F	35386	FS2	27/06/2009	Sub Acute Appendicitis	Lap. appendicectomy
10	Baby Rani	31/F	50530	FS2	10/09/2009	Calculous Cholecystitis	Lap. Cholecystectomy
11	Ambika	43/F	53375	FS2	24/09/2009	Calculous Cholecystitis	Lap. Cholecystectomy
12	Vijaya Lakshmi	21/F	49007	FS2	05/09/2009	Sub Acute Appendicitis	Lap. appendicectomy
13	Chandra	45/F	24807	FS2	07/05/2009	Calculous Cholecystitis	Lap. Cholecystectomy
14	ALAGU	40/F	9380	FS3	27/02/09	Calculous Cholecystitis	Lap. Hernia repair
15	Mohanapriya	14/F	53453	FS3	10/10/2008	Lap. Cholecystectomy	Lap. Appendicectomy

16	Selvi	30/F	22429	FS3	12/05/2008	Sub Acute Appendicitis	Lap. Appendicectomy
17	Asharu deen	16/M	21031	MS4	25/04/08	Sub Acute Appendicitis	Lap. Appendicectomy
18	Venu Gopal	30/M	19217	MS5	16/04/2009	Left inguinal hernia	Lap. TAPP
19	SUDHKAR	28/M	1435	MS4	18/01/08	Bubunocoele	Lap.hernia repair
20	SHANKAR	23/M	23937	MS4	10/05/08	Inguinal hernia	Laparoscopic repair
21	Ajesh	43/M	39407	MS5	06/08/2009	Hiatus hernia	Lap.Fundoplicatio
22	Sathiya seelan	14/M	6461	MS4/S3	09/02/09	Sub Acute Appendicitis	Lap. appendicectomy
23	Sivaswwamy	55/M	49010	MS5	03/09/2009	Right inguinal hernia	Lap. TAPP
24	Aaruswamy	42/M	33800	MS5	18/06/2009	Calculous Cholecystitis	Lap. Cholecystectomy
25	Vasanth	32/M	6571	MS4	22/02/08	RIF Mass	Diagnostic laparoscopy
26	Sundraj	53/M	31112	MS4/S3	08/06/09	Calculous Cholecystitis	Lap. Cholecystectomy
27	Selva kumar	42/M	15546	MS4	28/03/08	Umbilical hernia	Lap. Hernia repair
28	PALANI	45M	50504	MS4	10/10/08	Hydiad Cyst	Laparoscopy & proceed
29	Christo Clinton John	15/M	43175	MS5	08/08/2009	Sub Acute Appendicitis	Lap. appendicectomy
30	Mayil Samy	46/M	64662	MS4	01/12/08	Calculus Cholecystitis	Lap. Cholecystectomy

#### GROUP D

S.No	Name	Age/Sex	IP No	Ward	Date of Surgery	Diagnosis	Surgical Procedure
1	Gokila raj	49/F	5208	Special	01/02/2008	Calculous Cholecystitis	Lap. Cholecystectomy
2	Rathiya	29/F	8597	FS3	22/02/2008	Sub Acute Appendicitis	Lap. Appendicectomy

3	Bagiyalakshmi	25/F	23795	FS3	19/05/2008	Sub Acute Appendicitis	Lap. Appendicectomy
4	Ariya	14/F	44902	FS3	29/08/2008	Recurrent Appendicitis	Lap. Appendicectomy
5	Papathi	50/F	13057	Special	24/03/2008	Port site hernia	Lap. Hernia repair
6	Saraswathy	45/F	54772	FS3	24/11/08	Sub Acute intestinal obstruction	Diagnostic lap & adhesiolysis
7	SANGEETHA	20/F	72318	FS3	09/03/09	Sub Acute Appendicitis	Lap. Appendicectomy
8	Moorthyammal	55/F	50417	FS2	17/09/2009	Sub Acute Appendicitis	Lap. appendicectomy
9	Vijaya	45/F	10436	FS3	29/02/2008	Calculous Cholecystitis	Lap. Cholecystectomy
10	VALLIAMMAL	47/F	1185	FS3	02/02/09	Calculous Cholecystitis	Lap. Cholecystectomy
11	Kayalvizha	18/F	41942	FS2	30/07/2009	Sub Acute Appendicitis	Lap. appendicectomy
12	Karpakavali	23/F	77448	FS3	25/08/2008	Rt.inguinal hernia	TAPP
13	Yaseera	13/F	61854	FS3	14/11/08	Sub Acute Appendicitis	Lap. appendicectomy
14	Vijaya	25/F	37867	FS2	09/07/2009	Sub Acute Appendicitis	Larospic appendicectomy
15	Anjugam	42/F	50526	FS2	19/09/2009	Sub Acute Appendicitis	Lap. appendicectomy
16	Rajammal	45/F	54762	FS3	24/10/2008	Calculous Cholecystitis	Lap. Cholecystectomy
17	Vanitha	29/F	46145	FS2	22/08/2009	Sub Acute Appendicitis	Lap.

							appendicectomy
18	Gandhimathi	25/F	22209	FS2	09/05/2009	Sub Acute Appendicitis	Lap. appendicectomy
19	Bagiyam	17/F	26424	FS2	14/05/2009	Recurrent Appendicitis	Lap. Appendicectomy
20	Sakthivel	39/M	39382	MS4/S3	24/07/09	Sub Acute Appendicitis	Lap. appendicectomy
21	Yuvaraj	22/M	32402	MS5	11/06/2009	Acute Appendicitis	Lap. appendicectomy
22	Aarusamy	39/M	70074	MS4/S3	05/01/09	B/L inguinal hernia	Lap. TAPP
23	Sharman	55/M	9423	MS4/S3	23/02/09	Incisional hernia	Lap. Hernia repair
24	HARA HARAN	18M	47734	MS4	08/09/08	Indirect inguinal hernia	Lap.repair
25	MURUGAN ANTHAM	35/M	155	MS4	18/01/08	Calculous Cholecystitis	Lap. Cholecystectomy
26	RAJA	36/M	48489	MS4	05/09/08	Umbilical hernia	Lap. Hernia repair
27	Chandran	57/M	61730	MS4	14/11/08	Hepatoma	Diagnostic Laparoscopy
28	Durai	35/M	26329	MS5	21/05/2009	Calculous Cholecystitis	Lap. Cholecystectomy
29	Prakash	23/M	50510	Ms5	12/09/2009	Sub Acute Appendicitis	Lap appendicectomy
30	Rajarathinam	65/M	32385	MS5	23/07/2009	Right bubunocoele	Lap. TAPP